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Glyceryl trinitrate before coronary angiography

Sir,

The factual information in a recent publication by Dr Feldman *et al.* (Feldman, R. L., Pepine, C. J., Curry, R. C., Conti, C. R., 1978, *British Heart Journal*, **40**, 992–997) is an excellent contribution, I am concerned that the conclusions are philosophical and deviate from the practical aspects of evaluation of patients with suspected coronary artery abnormalities.

Abnormality implies deviation from the normal. Glyceryl trinitrate has been shown usually to dilate vasoactive coronary arteries from the diameter present before therapy. What is normal? Is the coronary artery calibre of an unpremedicated patient in an unfamiliar room, whose body is being 'invaded', more normal than the coronary artery calibre of a sedated, vasodilated patient?

Is the accuracy of measurement of a narrowed vessel with normal (or possibly enhanced) vasomotor tone as reproducible as in a dilated vessel?

Which measurement of narrowing or fixed obstruction with or without dilators and/or sedation would correlate best with clinical symptoms, operative findings, lactate abnormalities, or ventriculographic abnormalities?

My clinical impression is that the sedated, vasodilated patient can be accurately studied. The angiographic data are reliable guides to clinical status and operative intervention. Evaluation of the noncoronary dilated patient is of great value in scientific evaluation. Nevertheless, the withholding of coronary dilators will cause referrals for coronary artery bypass surgery of normal arteries that are vasoconstricted. The differentiation between organic and functional narrowing is never easy.

If coronary angiography is performed without coronary vasodilators, it should be repeated during the same study after adequate coronary vasodilatation.

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This letter was shown to Dr Feldman and his co-authors who reply as follows:

Sir,

First, we agree that it is difficult to know what is 'normal' with regard to coronary artery size and narrowing. To our knowledge, no one has specifically reported the influence of sedation on coronary artery diameters in man.

Secondly, in our experience, we have found that 'accuracy' of measurements of narrowed vessels of patients with presumably 'normal' vasomotor tone (i.e. without Prinzmetal's angina) are as reproducible as measurements made in vessels dilated by glyceryl trinitrate (Feldman *et al.*, 1979a). Our recent studies suggest that if measurements are made using a high-quality angiography system with an optical measuring device, a very high level of accuracy is achieved (Feldman *et al.*, 1979a, b). This level of accuracy (94 to 96%) probably represents the maximum possible with current angiographic systems.

Thirdly, there are no data that we are aware of, to relate symptoms, operative findings, lactate abnormalities, or ventriculographic abnormalities with measurements of fixed obstructions with and without coronary dilators or sedation. We can only speculate that the size of coronary arteries in the absence of drugs would correlate best with symptoms, etc.

Finally, we emphasise that the comment that the sedated vasodilated patient can be 'accurately' studied is only Dr Coskey's impression. Since signs and symptons of ischaemia occur only infrequently in sedated vasodilated patients, we take the view that angiographic and haemodynamic data are possibly distorted and do not reliably represent the patient's clinical status. We do not agree that evaluation of the non-coronary dilated patient is only of scientific value. In sedated patients or anaesthetised animals, glyceryl trinitrate (Gensini *et al.*, 1971) clearly increases the diameter of normal coronary artery segments. If a patient is studied only after glyceryl trinitrate, the investigator might miss an unexpected case of coronary spasm. Laboratories that routinely use sedation and vasodilators rarely report seeing coronary spasm in contrast to those laboratories that do not use these agents before angiography (Bennett, 1976). In addition, infrequently, a 'paradoxical response' to glyceryl trinitrate is observed (Feldman *et al.*,

1978), and Fig. 4, *British Heart Journal*, 1978, **40**, 992–997). We are unaware of published data to support Dr Coskey's contention that withholding coronary dilators causes referrals for bypass surgery of normal arteries. None the less, we emphasised in our manuscript that routine coronary angiograms should be performed before and after glyceryl trinitrate. We are not alone in this recommendation (Chahine and Luchi, 1976).

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